THE RATIONALE OF PORTACAVAL ANASTOMOSIS*

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SOME of the cirrhoses, certain of the hepatosplenopathies and many of the congestive splenomegalies have some common manifestations, usually evidences of long-standing chronic lesions.

Two symptoms that have always been dreaded, by patient and physician alike, are ascites and gastrointestinal hemorrhage. These are omens of serious pathology, usually associated with severe liver damage or portal hypertension or both. The ascites usually recurs after paracentesis, and once hemorrhage has occurred as a result of ruptured esophageal varices recurrent bleeding is common and sooner or later fatal. The dread of these symptoms by physicians is due to the fact that in the past no adequate therapy has been found and any new method that offers hope of dealing with these two symptoms deserves careful trial and evaluation.

The ascites is usually associated with liver damage and abnormal serum protein findings, especially a reversed albumin-globulin ratio. The severe hematemesis is usually from ruptured esophageal varices, the result of a portal hypertension, caused by either an intra- or extrahepatic portal block. In some of the cirrhosis cases less severe bleeding occurs into the gastrointestinal tract, over a period of days or weeks, resulting in a persistent anemia; the loss of blood is due to a persistent oozing from the mucosa of the intestinal tract associated with a prolonged prothrombin time, the result of a severely damaged liver.

In the patients with ruptured esophageal varices due to portal hypertension the portal block may be intrahepatic, in a cirrhotic liver, or extrahepatic with normal liver, and a congestive splenomegaly and distended portal radicals.

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It is obvious that any therapy to combat either one of these major symptoms or both must be preceded by a most careful study of the patient, to determine as far as possible the presence of a normal or a damaged liver and the presence of an intra- or an extrahepatic portal block. In such a study certain liver function tests as well as the physical findings, such as abdominal ascites, dilated superficial abdominal veins and cirrhotic facies, the size of the spleen, x-ray studies of the esophagus, are all important in differentiating intra- and extrahepatic lesions.

It is obvious that whether the portal block is intra- or extrahepatic the blood entering the liver by the portal vein is diminished in proportion to the narrowing of the diameter of the portal radicals. Nature provides a variable amount of shunting of portal blood to the systemic venous system by new dilated veins that develop from potential anastomotic veins in the abdominal wall, the retroperitoneal areas around the kidney and diaphragm, and between the veins around the esophagus and the azygos system and the hemorrhoidal plexus and the branches of the internal iliac veins. Any shunted blood has to pass through the systemic veins and lungs and reaches the liver with arterial blood through the hepatic artery.

Undoubtedly a relatively normal liver with extrahepatic portal block can utilize such by-passed blood more readily than a severely damaged cirrhotic liver, especially if some of the portal blood is still reaching the liver through narrowed intrahepatic portal channels. In the cirrhotic group the portal hypertension is usually not as high as in the cases with extrahepatic block, but because of abnormal albumin-globulin ratios ascites is more often present. In the patients with extrahepatic block with hypertension causing esophageal hemorrhage the shunting of the blood from the portal vein to the systemic system is more necessary and better tolerated by the liver, for there is less blood reaching the liver by way of the portal.

For many years attempts at shunting procedures have been tried with a sound rationale, but have proved generally unsuccessful because of the small vessels used in the shunting operations, the suture techniques used and the failure to use the supportive measures now available in the preoperative, the operative and the postoperative periods. These attempts have utilized branches of the mesenteric veins and smaller venous channels such as the ovarian and spermatic veins. The Eck fistula procedure was tried in a number of patients in France and Germany in

1910-1912 but with only one six months survivor. The cause of the prohibitive operative mortality was most frequently a renal shutdown, largely the result of shutting of the vena cava for too long a period during the suture anastomosis of the portal vein to the vena cava.

For many years one of our most discouraging follow-up problems in our Spleen Clinic at the Medical Center has been that of dealing with recurring hemorrhage in patients who had had a splenectomy for congestive splenomegaly or Banti's syndrome. Efforts at suture anastomosis of the smaller portal and systemic veins had been unsuccessful. We had encountered the problem of ascites and hemorrhage repeatedly without any promise of a successful therapy. But following Dr. Blakemore's brilliant results in bridging defects in large arteries and veins by means of direct anastomosis or by vein grafts, using vitallium cuffs to evert ends of the vessels to provide endothelium to endothelium anastomoses, this method offered definite promise in uniting the larger trunks of the portal system to those of the vena cava and ultimately of carrying out an Eck fistula in selected cases.

These portacaval shunt operations are very difficult and are still in the experimental stage and Dr. Blakemore, Dr. Humphreys and I have not done enough of them or followed the cases operated upon long enough to make any positive or dogmatic statements. But certain of our patients have shown some unexpectedly good results and we have been encouraged to attempt more of these procedures in a group that are otherwise doomed. What is most important from now on is to determine more definitely the best procedure for the patient with intra- or extrahepatic block, and to establish better criteria for differentiating the various lesions giving rise to the arresting symptoms of ascites and gastrointestinal bleeding.

Dr. Hanger will discuss the latter problem and Dr. Blakemore will present the technical operative problems and the results thus far observed. This presentation is from the Combined Spleen Clinic in which a team of internists, pathologist and surgeons have been coöperating.